XVIIth International Congress of Medicine
London: 1913

Hygiene and Preventive Medicine

The Factors that determine the Rise, Spread, and Degree of Severity of Epidemic Diseases

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LONDON
HENRY FROWDE
OXFORD UNIVERSITY PRESS
HODDER & STOUGHTON
WARWICK SQUARE, E.C.
1913

SECTION XVIII

HYGIENE AND PREVENTIVE MEDICINE

DISCUSSION No. 3

THE FACTORS THAT DETERMINE THE RISE, SPREAD, AND DEGREE OF SEVERITY OF EPIDEMIC DISEASES

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Part I

(*a*) *Introduction*

It is usual to regard an epidemic disease as a malady which affects a number of human beings either simultaneously or within narrow limits of time, and the classical definitions either assert or imply that the chief part in the causation of such diseases is played by something external to the sufferer. This conception is illustrated in the tractate Περὶ φύσιος ἀνθρώπου (Van der Linden's edition, i. 274; Fuchs's translation, i. 203), which is frequently included among the writings of Hippocrates, although its authenticity is doubtful, while the same ideas are embodied in a definition from Sydenham's *Medical Observations*, where we read (I. i. 6):

'Acutos quod spectat (quos impraesentiarum tractare mihi est animus) eorum alii a secreta atque inexplicabili aeris alteratione, hominum corpora inficientis, gignuntur, neque a peculiari sanguinis et humorum crasi omnino dependent, nisi quatenus occulta aeris influentia dictis corporibus eandem impresserit. Hi, durante arcana illa aeris constitutione nec ultra, pergunt lacessere, neque alio ullo tempore invadunt; *Epidemici* hi dicti sunt.'

Sydenham does not so specifically repudiate the intervention of a personal factor as did the Greek author, who regarded the cause as wholly extrinsic.[1]

[1] My friend Professor Sticker points out to me that Fuchs's translation of the relevant passage is not quite exact, and I owe to him the following version: 'Wo aber eine einzelne Krankheit als Volkskrankheit herrscht, da ist selbstverständlich nicht die Lebensweise daran schuld, sondern die Luft, die wir einatmen, ist schuld; und auch dieses ist klar, dass die Luft dabei eine krankmachende Ausscheidung tragen mag.'

Although the classical definitions assign to the external factor a principal rôle, it need not be concluded that their authors believed that, so far as epidemic disease is concerned, 'all things come alike to all,' and we shall be departing but little from the ancient standpoint, while taking up a position consistent with modern practice, if we define an epidemic as an outbreak of disease affecting a number of persons simultaneously, or within a short interval of time, and such that all the attacks, however modified by individual peculiarities, present features of general similarity. The laws which may be deduced from a survey of epidemic diseases as experienced now and in the past, and appropriate to describe their origin, rise, and fall, form the subject of this discussion. It is my duty as one of the reporters to indicate certain of the methods of inquiry and results which seem worthy of consideration. I am under no illusions as to the magnitude of the problem, and must at the outset claim the indulgence hinted at in the words 'in magnis voluisse sat est'.

(*b*) *The Classical Conceptions*

A scientific description of epidemic diseases, i.e. an epidemiological law, presupposes on the part of its discoverer: (1) the possession of a disciplined and creative imagination, the mysterious spirit which the Hebrew poet conceived as moving over the face of the waters, fashioning therefrom an ordered creation; (2) a wide clinical experience of disease; (3) a considerable knowledge of contemporary and antecedent medical history. Two of these conditions were fulfilled by our greatest predecessors, by such men as Hippocrates, Avicenna, and Sydenham, but the third no less essential requirement was not and could not be satisfied. A very superficial acquaintance with epidemiological history is enough to demonstrate that the experience crowded into even the fullest of human lives will not be sufficient to provide data for the scientific description of epidemic disease. If we think merely of a single type of record, morbidity statistics, we shall admit that even now the available information falls short of the ideal, but that its superiority to anything extant even so recently as the seventeenth century is enormous.

It follows that we ought not to expect any complete epidemiological theory in the classical literature of our science, and we do not in fact find any attempt to formulate such a theory in the writings of our greatest predecessors. Hippocrates studied in masterly fashion the connexion between terrestrial phenomena and morbidity, and in his *Epidemics* laid down a system of collecting and arranging data for the use of some future synthesist which was copied by his greatest successor in the field; but it cannot be said that the father of medicine left us a body of epidemiological theory.

In the centuries which part Hippocrates from Sydenham but little progress was achieved. The principal authors, however much they might differ as to its detailed application, subscribed to the doctrine that epidemics were mainly due to an extrinsic cause. Their speculations

as to the nature of this cause and its mode of action, often ingenious, sometimes profound, are of importance to the historian but need not detain us here.[1]

Sydenham's contributions to the subject must be noticed with more attention, and from the *Medical Observations* we can reconstruct a fairly complete account of the epidemiological principles of that great observer. Sydenham had no doubt as to the difficulties involved in the task of framing a theory of epidemics. Thus he wrote :

'Quin et magis adhuc (si fas est dicere) impossibile fuerit variorum Epidemicorum species tradere, qui a specificis aeris alterationibus oriuntur, quantumvis in proclivi illud esse videatur, istis videlicet qui *febrium* nomina attexere norint notionibus, in speculatione istarum alterationum quae in sanguine humano atque eius humoribus per hanc illamve principiorum degenerationem fieri possunt, male fundatis.' [2]

He held, indeed, that the ætiology of epidemics might be in truth inexplicable ; [3] but, while rejecting merely subtile speculation as being of as much use in medical treatment as music in bricklaying,[4] affirmed his unshaken belief in the orderly succession of phenomena.[5]

Sydenham emphasized the necessity of studying the true and authentic features of an epidemic in a uniform disease,[6] commented on the variations which occurred in the same disease,[7] remarked the tendency of particular types to prevail at certain times,[8] and definitely disclaimed an attempt to refer specific epidemics to corresponding changes in the atmosphere.[9] Perhaps the most significant utterance is the passage which deals with the secular phenomena of disease. After giving reasons for a belief that small-pox was unknown in the time of Hippocrates, Sydenham writes : [10]

'Quocirca opinari mihi fas sit, morbos certas habere periodos pro occultis illis atque adhuc incompertis alterationibus quae ipsius terrae accidunt visceribus, pro varia scilicet eiusdem aetate ac duratione ; quodque, sicuti alii morbi iam olim extitere, qui vel iam ceciderunt penitus, vel aetate saltem pene confecti exolevere, et rarissime comparent (cuius modi sunt lepra atque alii fortasse nonnulli), ita qui nunc regnant morbi aliquando demum intercident, novis cedentes speciebus, de quibus nos ne minimum quidem hariolari valemus.'

We may summarize the teaching of Sydenham as follows : an epidemic disease should be regarded from three points of view : (1) There is a process

[1] Among writers greatly studied in mediaeval and early modern times who dealt with the subject, we may cite (1) Actuarius, *De Methodo Medendi*, who, in book v, speaks of (Stephans's edition) 'Omnes pestilentes morbos, quique populatim grassantur, coeli vitio enatos'. (2) Aetius, *Tetrabib.* II, Sermo i, cap. 95. (3) Avicenna, *Liber Canonis*, book iv, tract. 4 (where will be found the famous reference to rats in connexion with pestilence) and *De Removendis Nocumentis*, tract. ii (Alpagus translation, edition 1557, Venice). Of the various theories of external causation, perhaps those of Paracelsus (see Haeser, ii. 93) are likely to appeal most to the modern reader.

[2] *Observationes Medicae*, i. 2, 22.

[3] Ibid. i. 5, 5. [4] Ibid. ii. 2, 47. [5] Ibid. ii. 2, 48. [6] Ibid. i. 2, 7.

[7] Ibid. i. 2, 9. [8] Ibid. i. 2, 15. [9] Op. cit. i. 2, 22. [10] Ibid. v. 4, 16.

of secular or long-period modification in virtue of which a specific type becomes dominant in a particular epoch and then gradually or suddenly gives place to a rival. (2) There is another set of factors producing short-period oscillations in the epidemicity of a given disease leading to the phenomena of seasonal prevalence. But, finally, we can connect these two trains of ideas by the conception of an epidemic constitution in virtue of which certain types of epidemic or certain features of morbidity tend to prevail at a given time to the exclusion of other types or other symptoms. We may say that the amplitude of the first kind of wave is measured in centuries, that of the second in months, and that of the last in years.

This analysis is, of course, imperfect, particularly in its attempt to characterize so intricate an idea as that embodied in the doctrine of epidemic constitutions, but the antithesis between the secular and ephemeral aspects of epidemic disease does, I think, correspond to realities worthy of our close attention. Just as the student of geology must combine a knowledge of the changes now proceeding in the crust of the earth with an investigation of those effected in past time to make a complete science, the epidemiologist cannot hope to grasp the whole truth if he concentrates his attention upon contemporary or recent phenomena. One should, I think, endeavour to follow the vicissitudes of some definite disease through a considerable period of time in order to realize the existence of secular factors, and then separately investigate the immediate features of various epidemics. These two lines of study are not, indeed, absolutely distinct, yet little is lost and something gained in respect of clearness if we agree so to regard them for the purposes of description. I propose to follow this order in my subsequent remarks, and hope to have made it seem probable that such a course is in accord with the views of our greatest English epidemiologist.

The first branch of the inquiry can be most conveniently illustrated by the case of plague, but as this is a subject which my colleague, Professor Dr. Sticker, can treat with far greater knowledge than I, it will be better to confine myself to epitomizing the history of scarlet fever, a less satisfactory but still instructive example.

(*c*) *Secular Changes in Epidemicity as illustrated by Scarlet Fever*

That scarlet fever prevailed in ancient times is a proposition we can neither prove nor disprove, and although we shall probably agree with Haeser (iii. 69) that some proportion of the disease termed morbilli by the translators of the Arabian physicians may have been scarlet fever, it is equally impossible to reach any satisfactory conclusion as to its prevalence or fatality in mediaeval times. This is particularly tantalizing since were morbilli to any extent identifiable with scarlet fever the latter may have been more fatal than small-pox in the Middle Ages.[1]

[1] Schnitzlein argued that scarlet fever was known to Rhazes, and Willan also contended for its antiquity. A careful discussion will be found in Richter's paper.

The modern history of scarlet fever may be said to date from Ingrassia's account of a Palermo epidemic disease towards the end of the sixteenth century (Haeser, 421 ; Hirsch, 171 ; Richter, 176). For the seventeenth century, Most tables no less than fifteen epidemics (Most, i. 137), but some of the identifications are, to say the least, conjectural. Up to the end of this century the evidence is indeed so scanty that the most we can hope to gain is that unsubstantial thing, a general impression. The impression of that character which I have personally formed is that the disease was uncommon, this being correlated with the reigning pestilential constitution and perhaps also the rising prevalence of small-pox, but that when it did occur the fatality was considerable, as suggested by the words of Sennert and Winsler (see Haeser, iii. 423). We reach firmer, if still treacherous, ground in the closing years of the century.

The *loci classici* in the writings of Sydenham and Morton are too familiar to need citation ; I would merely remark that while these passages suggest that Morton had, and Sydenham had not, observed malignant cases with anginal complications, the conclusion that this may be referred to the different social strata from which the two physicians' patients were drawn is hardly confirmed by the teaching of modern experience. It should also be remembered that Sydenham's dislike of fussy medical interference may have predisposed him to under-rate the gravity of the disease. Whatever may have been the rate of fatality, it seems clear that the prevalence was slight up to the second decennium of the eighteenth century. There are indications that between 1716 and 1732 the prevalence was considerable but the fatality low. From 1740–60 a graver disease confronts us, but there is some dispute as to how far the epidemics were anginal scarlet fever, and how large a part was played by true diphtheria (particularly in the famous New England epidemics). The most familiar description, at least to English students, dating from this epoch is that of Fothergill, who described an epidemic sore throat which was, he says, severe in and around London in 1746, attacking more females than males, rare and non-fatal in healthy adults. It is plain from a remark of Fothergill's (cited by Creighton, ii. 697) that he did not believe the disease to be Sydenham's scarlet fever. On the other hand, Creighton justly directs attention (ii. 711) to the difficulty Withering and others have experienced in distinguishing what they took to be true *Scarlatina anginosa* and the *Angina gangrenosa* of Fothergill.[1] We may, perhaps, regard this period as one of moderate epidemicity and high fatality. From 1750–70 a period of increasing epidemicity, especially after 1760, is traceable. Within these termini Most records eleven epidemics : five of these (Switzerland 1762, Copenhagen 1762, Halle 1764, France 1765, Westphalia 1769–70) seem to have been mild ; two (France 1762, Holland 1769–70) malignant ; the others doubtful.

[1] Rumsey in his account of an epidemic sore throat at Chesham in 1788 describes what would now be held to be a typical scarlet fever outbreak, but held the throat symptoms to be of primary importance and the rash not to be pathognomonic.

From 1770 onwards our knowledge becomes increasingly definite, and we owe to such writers as Rumsey, Withering, Wedemeier, and Zimmermann good descriptions of epidemic scarlet fever, unfortunately not illustrated by statistical records. It is patent that towards the close of the century a decided increase in malignancy was observed which is alluded to in the well-known remarks of Graves on change of type. Most especially remarks the severity of the disease between 1799 and 1801, and there is much evidence of malignant scarlet fever in London about this time (Willan and others). By 1803 the disease was less prevalent in Germany and, after a bad year in 1811 (Most), there are signs of a general remission, the tide not rising again until the years immediately preceding 1820. The scarlet fever of this time called forth several excellent papers such as those of Berndt and Zierl. A few statistics of this period are available which *suggest* that although the prevalence was increasing the malignancy was not great.

TABLE 1

Years.	*Town.*	*Population.*	*Scarlet Fever Deaths.*	*Authority.*
1819	Stadthagen . .	7,200	34	Most
,,	Bückenburg . .	6,000	26	,,
,,	Hagenburg . .	4,500	12	,,
1821	Hamburg . . .	130,000	212	,,
1817–18	Custrin . . .	30,000	201	Berndt

TABLE 2

CASE MORTALITIES IN A FEW OUTBREAKS

Years.	*Cases.*	*Deaths.*		*Source of Data.*
1777–9	146	18	12·3 per cent.	Newcastle Infirmary (Creighton)
1779–89	57	8	14·0 ,,	,, ,, ,,
1790–92	152	7	4·6 ,,	,, ,, ,,
1803	171	7	4·1 ,,	Ackworth School (Creighton)
1817–18	1,234	201	16·3 ,,	Custrin (Berndt)
1803–27	795	30	3·8 ,,	Newcastle Infirmary (Creighton)

During the third decade, especially between 1824 and 1828, scarlet fever was severe on the Continent, but it is not until 1831 that signs of the malignancy that characterized the disease at the beginning of the century reappear in English epidemiological literature. The severe epidemic of 1834 in various parts of Ireland gave occasion to Graves for the utterance of his warning as to the danger of attributing variations in fatality to methods of treatment (Graves, i. 304). This return of malignancy to a disease become generally prevalent is one of the noteworthy features of the subject. After 1837 the system of general registration in England renders information, at least ostensibly, more precise, and, following on a great increase of the deaths in 1840, scarlet fever ' continued year after year for a whole generation to be the leading cause of death among the infectious maladies of childhood ' (Creighton, ii. 726).

Since the decennium 1861–70, the mortality per mille from scarlet fever has declined, nor can the fall be attributed in any large measure to the undoubted transference of cases formally classified as malignant anginal scarlet fever to the heading diphtheria [1] (Diagram 1).

We have now reached an epoch in which the improvement in statistical records enables the inquirer to render a tolerably precise account of the changes in scarlatinal prevalence and fatality so far as certain areas are concerned. This statement applies to the administrative county of London, from which, owing to the operation of the Notification of Infectious Disease Act, we can obtain a record of the great majority of cases since 1892.

In Tables 3–5 [2] will be found the principal data relative to London, and in Table 7 the crude fatality rates of the city of Hamburg (the figures up to 1903 are cited from Prinzing, the remainder from the *Hamburg Medizinalberichte*). It is, however, difficult to effect international comparisons without more detailed knowledge of the registration systems than can be gleaned from official reports. For instance, in the city of Berlin, apart from changes in nomenclature used on notification papers (e.g. in 1900, 'Bösartiger Scharlach' seems to have been used instead of 'Scharlach'), it is plain from Table 6 that the system of notification was incomplete. In view of the fact that the fatality of scarlet fever is variable, the more exact mortality figures do not give us all the information we require. We are forced to abandon an attempt to study geographical variations of prevalence and fatality in civilized countries.

I shall confine myself to asking whether the fatality and prevalence of scarlet fever have changed in London during the past twenty years. In his study of scarlet fever in Denmark, Heiberg calls attention to the necessity of caution in drawing conclusions from the official statistics of this disease. There is a danger when we have returns taken over a long series of years, during which not only the stringency of enforcement of a notification law but also the diagnostic criteria may have changed, lest we compare things not truly comparable. It follows that conclusions drawn from serial statistics must be accepted with reserve.

But while we recognize these limitations of our material, we must not less clearly recognize that the statistical method, with all its imperfections, is the only one by means of which this problem can be elucidated. With these warnings, I return to the London figures. First as to the fatality. It will be noticed that the case mortality has diminished with some regularity. A consideration of the details shows that the change has been almost confined to the ages below ten, but, in view of the large numbers, and assuming—as is probably justifiable—that no systematic error vitiates the returns, we can hardly doubt that the change is a

[1] For the relations between scarlet fever and diphtheria, see the papers of Longstaff and Ransome.

[2] The remaining tables are collected at the end of part ii. Some of the diphtheria statistics are included for the sake of comparison. I have to thank Dr. W. H. Hamer for kindly supplying me with the London data.

significant one. Our conclusions respecting prevalence are decidedly less definite. Tables 8 and 9 are an attempt to provide further material for study, but their value is not of a high order. Apart from the fact that the population statistics are not accurate for intercensal years (the total populations were estimated by the usual methods, but this process has not been applied to the populations at ages which were assumed to be those of the foregoing census year), the ratio of total cases to total population within a huge heterogeneous area like that of the administrative county of London may be misleading.[1] Taking the figures simply as they stand, they suggest that there has been some diminution in prevalence.

We need some method by means of which we can compare the respective diminutions in fatality and prevalence reduced to a common scale. A first approximation to such a method is afforded by using the coefficient of correlation. Thus, if we correlate fatality or prevalence with time, a negative value will indicate that the fatality (or prevalence) has diminished as we approach the present day and the relative magnitude of the two coefficients will be some measure (of course but a rough one) of the respective intensities of the associations. I find, that the correlation of fatality with time is $-\cdot85 \pm \cdot04$ and of prevalence and time, $-\cdot52 \pm \cdot11$, the difference being nearly three times the 'probable error'[2] of the difference. In other words, the association of diminishing fatality with time is closer than that between diminishing prevalence and time, although naturally this statement is made with reserve when we reflect on the short series of years available for study. The general conclusion seems to be that while probably scarlet fever is becoming both less prevalent and less fatal, the latter change is decidedly more distinct than the former. What we really need is a collection of statistics from a wider area and extending over a longer period of time, and it is satisfactory to know that a report similar to a recent publication of the English Local Government Board [3] will become an annual return.

Let us now consider the possible causes of the effects just displayed. If in any population the death-rate of a given disease falls faster than the death-rate in general, either some new disease has come into existence or an extant disease has improved its position in the struggle for pre-eminence as a factor of mortality. This truism has not been sufficiently reflected upon by the lay enthusiasts who seem to regard the diminution or disappearance of any disease as of necessity a pure gain to the public. The first writer

[1] In Table 10 I provide *fatality* data for one of the metropolitan boroughs (taken from the report of the medical officer of health for Poplar), and it is in general agreement with the figures for all London.

[2] The 'probable error' calculated by the usual formula is known to be of doubtful value in such cases as the present, and the correlation is not true variate correlation as one of the characters, years arranged in series, is not a true variable but an arrangement in 'ranks'.

[3] *Statistics of the Incidence of Notifiable Infectious Diseases in each Sanitary District in England and Wales during the year* 1911 (L.G.B. Reports on Public Health, &c., Subjects, New Series, No. 64).

to illustrate this Principle of Substitution, as it has been termed, was Robert Watt, of Glasgow. Watt showed that, notwithstanding an enormous diminution in the proportional mortality of small-pox between 1783 and 1812 at Glasgow, the ratio of deaths under ten years to total deaths had actually increased. He further showed that the relative mortality of measles had increased as that of small-pox decreased; thus, the latter fell from 19·55 in the first period of six years to 3·90 in the last, the former, on the other hand, increased from 0·93 to 10·76. Watt remarks: 'The great increase of deaths between two and ten years of age is very remarkable. In the first period they amounted to no more than 14·08 per cent.; in the last period they come little short of 20 per cent. Are we to expect a continuation of this increase of deaths from ten to fifteen, generally a very critical period of life; and in the ages from fifteen to twenty? As matters now stand, we have gained under two; we have lost between two and five, and also between five and ten. At ten we stand nearly on the original level, but if we are to lose between ten and twenty, it shows us how truly abortive all our schemes have been. We may, it seems, by the permission of Divine Providence, deprive death of some of his apparently most efficient means, but deprived of these, new means are discovered, or the old improved' (Watt, pp. 380–1). Since Watt does not seem to have compared the deaths with the populations living at various ages, his work only deals with the relative shares of the diseases so that his results may be consistent with a great absolute saving of human life in the period with which he dealt. Fifty years later, Farr called attention to Watt's results, pointing out the then growing importance of scarlet fever and diphtheria as absolute factors, and also when compared with small-pox. Farr invoked the principle of selection to elucidate the process, writing: 'For if there is a struggle for existence among the visible forms of life, and if the struggle is the severer, the nearer these forms are allied, is there not the same struggle among the elementary independent particles of life to which epidemics are due?' (Farr, p. 325). In our own time, Creighton has characterized Watt's conclusion as 'one of the soundest and most instructive generalities in epidemiology' (Creighton, ii. 656).

In Table 11 I have collected some of the official figures illustrating the nature of the substitution during the last fifty years in England and Wales. Relative values were obtained by dividing each entry in columns 2–5 by the deaths from all causes for the same year or decennium. It will be seen that no single exanthem occupies the old position of scarlet fever as an absolute factor of mortality, but that the relative importance both of measles and whooping cough has tended, with irregular variations, to increase. It is, however, to be observed that not one of these diseases shows any distinct absolute increase; there is no question, therefore, of *absolute*, but only of *relative* substitution. Finally, I must glance at the explanations which might, on theoretical grounds, be put forward to describe the facts.

The layman would unhesitatingly advance the following explanation. *The absolute and relative diminution in scarlet fever mortality is the result of interference with the disease ab extra, consequent upon improvements in sanitary administration and clinical methods.* Our attitude towards infectious disease has been modified in consequence of (*a*) improved facilities for and increasing stringency of isolation ; (*b*) improvements in the actual treatment of cases ; (*c*) improvements in the art of diagnosis.

It may, I suppose, be said that (*b*) and (*c*) have not operated particularly in favour of scarlet fever as compared with other exanthems. On the other hand (*a*) will naturally exert the greatest effect upon the most contagious malady and has, in addition, been rigidly applied to scarlet fever and diphtheria alone among the diseases here considered.

But we must bear steadily in mind the fact that this problem is more intricate than the layman supposes. All competent observers do not seem to be agreed as to the true bearing of isolation upon the general scarlet fever mortality rate. Wilson, for instance, in a careful study published some years ago, argued that the lessened importance of scarlet fever was attributable rather to diminished fatality than to lessened prevalence. He pointed out that the fall was apparent before modern administrative methods were in generally effective operation, and published data which were consistent with little if any change in prevalence. I have already pointed out that, although there is evidence of diminishing prevalence in the county of London, it is less strong than that in favour of a lessened fatality.[1] With respect to the latter, it is worthy of remark that the changes are by no means uniform at all ages, thus the fatality at 10–15 has diminished little if at all. The less reliable prevalence statistics are also irregular. Sir Shirley Murphy has directed attention to variations in age incidence, and has observed that the aggregation of children in elementary schools may be an important factor. The suggestion is that the *materies morbi* may have undergone a change in infective power, with the result that children tend to pass through the earlier years of life unscathed, the disease only being generated at a period when opportunities of infection are multiplied, perhaps by attendance at school. A consequence might be that the body is only called upon to combat the disease when its physiological means of defence have been matured, but that the resulting diminution in fatality is partly neutralized by the survival of susceptibles from the earlier age-groups. I shall not enlarge upon these ideas, which, however, serve to indicate the difficulty associated with an attempt to measure the importance of any agent as a factor in producing changes of epidemicity.[2] We must, indeed, be cautious lest

[1] There can be little doubt, as has been pointed out to me by my friend Dr. R. S. Ewart, that a considerable number of mild attacks escape notification.

[2] For a fuller discussion of some of these points see the Forty-ninth Annual Report of the Registrar-General, p. xiv, &c., the Papers of Whitelegge, Ballard, and Murphy, also the Annual Reports of the Medical Officer of the Administrative County of London, where much valuable statistical information is to be found.

we fall into the error of Graves's teachers in the early nineteenth century.

Some have attempted to account for the facts on the basis of an appeal to natural selection. Scarlet fever was once widely prevalent, and it is possible that absolute or relative immunity depends upon innate heritable characters. Consequently the proportion of insusceptibles in a population tends to increase. If, however, the causative agent be an obligatory parasite, a diminution in the number of susceptibles necessarily involves a diminution in the number of opportunities to become infected. This, in its turn, leads to a slackening in the intensity of selection; innate immunity ceases to be a character of survival value and the way is paved for another formidable outbreak if only the disease have not become really extinct. We might suppose that the immunity acquired by the selective action of a series of bad epidemics is gradually lost, and that in a few generations the soil is ready for a fresh crop of the disease.

It is, of course, true that nothing in the ascertained history of scarlet fever disproves this hypothesis, but if one considers the vicissitudes of such a disease as plague through a very long period, as narrated with such wealth of illustration and lucidity by Professor Sticker, we shall have doubts as to the sufficiency of so facile an interpretation. We must not confine our application of the selection hypothesis to the human subject of the disease. Much work, in particular the researches of Penfold, Müller, and other bacteriologists, has demonstrated that mutations or even fluctuating variations may lead to the evolution of bacterial species, or perhaps, more accurately speaking, to the production of varieties differing profoundly from the parent stock in physiological properties. The application of these results to the phenomena of secular variations of epidemicity is a matter for the future in the case of a disease like scarlet fever, the *materies morbi* of which elude our actual powers of identification.

If we apply the selection hypothesis to the morbific organisms themselves, it seems clear that any obligatory parasite must either submit to modifications of its virulence, acquire powers of enormous multiplication, or perish in the struggle for existence. Here again we must remember the complexity of the problem. It is not only changes in the essential *materies morbi* of scarlet fever which are in question, but also variations in the secondary invaders which are so often responsible for the graver complications of the disease. In this connexion we should have to consider variations and mutations of myriads of pathogenic streptococci and require to take account of bacteriological phenomena which I am not in a position even to enumerate.

In an interesting paper by Nash and the comments thereon of the late Dr. Payne, the conception of evolutionary change among competing organisms of disease is traced out in some detail. It would exceed the functions of a reporter were I to attempt a critical examination of such work. The evidence as yet produced is hardly sufficient to enable

us to assign a very definite share in the reduction of scarlet fever to the action of the causes, or supposed causes, last enumerated. I will therefore simply conclude this section of my report by summarizing the conclusions which may be usefully debated in a discussion of secular variations of epidemicity.

1. Secular variations are too striking to be attributed wholly to errors of record, although in the particular case of scarlet fever the early history of the disease cannot be separated from that of diphtheria.

2. Such variations may be due to the action of natural selection operating upon variations of human resisting power, relative immunity being an inborn heritable character, or upon the biological characters of the causative agent, or by a combination of both processes together with some change in the characters of secondary parasitic invaders.

3. It is not permissible to attribute the totality of secular changes to the factors scheduled under 2. The direct action of public health authorities may have played an important part in bringing about the decline in prevalence and fatality of the disease.

4. Our present data are not sufficient to permit us to adjudicate upon the claims of the different possible factors. In particular we are not entitled to regard such diseases as scarlet fever, which at present are comparatively small factors of mortality, as extinct volcanoes. It is impossible to say that the experience of Graves in the nineteenth century will not be ours in the twentieth.

Part II

The Immediate Course of an Epidemic

That an outbreak of epidemic disease is an event fulfilling definite laws in respect both of its temporal and spatial evolution seems to be a conception grasped in its entirety only in modern times. That certain seasons of the year favoured pestilence, and that the lines of spreading were not random are, indeed, observations or deductions dating from a remote antiquity. The idea of an epidemic constitution, of a common bond of union between simultaneously prevalent maladies or of their assimilation to a dominant type, implies a recognition of order in the sequence of events, but is rather intended to account for the inter-relations of different diseases than to interpret the sequence observable more or less distinctly in each outbreak, the rise and decline of a specific pestilence. Further, the inherent vagueness of the conception rendered it susceptible of a variety of interpretations which have not conduced to a clear understanding of the fundamental problems.

I think, therefore, that it will facilitate discussion if I forgo any attempt to trace in chronological outline the history of opinion respecting the immediate phenomena of an epidemic and confine myself to a brief *résumé* of some significant points.

Sufficient attention has been devoted in the first part of this report to

secular changes in epidemicity for it to be merely necessary to repeat that the study of an epidemic without reference to the previous history of the disease involved can but lead to imperfect apprehension of the facts.

I shall now examine the immediate course of a few epidemics and in doing so, two aspects of the matter suggest themselves.

In the first place, an epidemic often, but not always, seems to be kindled at one or two foci within a populated area and then to spread in a regular fashion within the surrounding territory. The investigation of laws describing such dissemination may be termed the Spatial Theory of an Epidemic. In the second place, if we fix our attention upon an area without discriminating the elements of which it is composed, and consider the frequency of cases occurring within equal intervals of time, a regular association between the number of cases and the interval which has elapsed since the outbreak occurred can often be discerned. To account for this is the province of a temporal theory of epidemics. Fairly ample materials for the study of spatial dissemination are provided, principally in the reports of official investigators and commissions of inquiry, the most exact and detailed being, perhaps, those of the Indian Plague Commission in the Punjab. The scope of these inquiries and the necessity of bearing in mind minute peculiarities of local conditions render it impracticable to deal with them in the course of such a paper as this. I shall therefore pass at once to the second of the topics proposed, viz. the temporal theory of an epidemic. The logical order to pursue would seem to be first to analyse collections of data, reducing the irregular objective figures to continuous forms, which can be more readily grasped by the mind and used as the basis for deductions, by the aid of some method uniformly applicable to each and every case. In a very large number of cases the time graph of an epidemic regarded as a frequency polygon, the number of cases being plotted as ordinates and the intervals of time as abscissæ, suggests that the prototype is a monomodal frequency distribution. This fact (together with the further observation that the rise or fall of the epidemic curve, looked at as a function of the time, is often such as to suggest high contact on the part of the frequency curve at one or other, or both, ends of the range with the base line) would lead the statistician to think that Pearson's family of frequency curves, fitted from the moments of the real distributions and classified in accordance with the values of certain momental functions, might fitly be used in the study of epidemics.[1]

The justice of this view was, I think, first appreciated by Brownlee,[2] to whom statisticians and epidemiologists are indebted for several important contributions to the subject under notice. Brownlee analysed thirty-three epidemics by Pearson's method and found that all but three gave the momental constants required by a curve of Pearson's fourth type. In

[1] See note A.

[2] Contemporaneously Sir Ronald Ross was engaged in the development of a mathematical method, some remarks upon which are to be found in note B.

many of these cases the asymmetry, as indicated by the value of β_1,[1] was slight, but in only one instance were the values of β_1 and β_2 close to those required by the 'normal' curve, viz. 0 and 3. The instance in question was that of zymotic diarrhœa in Manchester, 1878–87, the time interval being one month, and the frequency distribution that of cases; the constants were, $\beta_1 = \cdot 0006$ and $\beta_2 = 2 \cdot 99658$. More recently I have applied the same process to epidemics of plague in certain Punjab districts in the course of an endeavour to answer the following questions: (1) Does the form of the epidemic time curve vary from one district to another? Is there any difference between the form of the curve describing a severe and that representing a mild epidemic? I used the deaths officially reported in the severe epidemic of 1906–7 in Amritsar district (23,947 deaths), in the relatively mild epidemic of 1905–6 in the same district (6,061 deaths), and in the severe outbreak of 1906–7 in Gujrat district (55,245 deaths). The curve appropriate to the Gujrat data was of type iv, markedly skew ($\beta_1 = \cdot 798$, $\beta_2 = 4 \cdot 781$, skewness $= \cdot 344$), a fair graphical representation of the data, but a poor fit when tested algebraically. The curves found for the other data were also markedly skew (Greenwood, p. 96). I have found the same marked skewness when dealing with more accurate figures derived from a circumscribed area. For instance, analysing the death returns from Poona city for the epidemic from July 1911 to March 1912, arranged at monthly intervals, the following constants were deduced, $\beta_1 = \cdot 107$, $\beta_2 = 2 \cdot 823$, skewness $= \cdot 212$. The obvious objection to following strictly the method just outlined is that a reduction of numbers of epidemics relating to different diseases or even to the same disease in different places or in the same place at different times sufficient to permit hopes of revealing the factors of epidemicity common to all of them, would be a task of enormous magnitude, since the analysis of even a single epidemic by this process demands some little time and care. Apart from these last considerations, which should not deter one from any attempt to arrive at the truth, if we bear in mind the very diverse circumstances which may be of importance for the temporal evolution of different epidemic diseases, it seems improbable that we should readily arrive at results so plain and distinct as to be unobscured by the host of minor causes proper to each disease and each locality. The report of the Indian Commission on the seasonal prevalence of plague in India (*Journal of Hygiene*, viii. 266) illustrates some of these difficulties very well. It is accordingly desirable to take a preliminary survey of the factors which might be operative and have been suggested by other methods of inquiry.

If we omit from consideration certain epidemics of functional nervous manifestations, such as the mediaeval Tanzwuth, we may lay it down that the realization of an epidemic demands three essentials, viz., an infectable subject, an infective object, a favourable environment.

[1] A more accurate test is to put the skewness equal to $\frac{\sqrt{\beta}\,(\beta+3)}{2(5\,\beta_2 - 6\,\beta_1 - 9)}$, but the value of β_1 is a sufficient indication for the present purpose.

(*a*) *The object* is in general organized ; under constant environmental conditions it will tend to pass through a cycle of changes expressing themselves by means of different effects upon the subject, and these changes may be hastened or retarded by variations of the common environment.

(*b*) *The subject*, being necessarily a living creature, is, *mutatis mutandis*, susceptible of changes congruent with those just predicated of the infective object.

(*c*) *The environment* must be understood to comprise not merely the direct reactions of, say, climatic or economic conditions upon the subject and object, but also the effects of the morbid evolution upon both together with the results of interference *ab extra*, for instance, in response to the activities of sanitary administrators.

(*d*) The investigator who has attributed a chief place to modifications of the infective object among the possibly influential factors is Brownlee. This author describes the ways in which an epidemic may be brought to an end in the following terms : ' Firstly, the conclusion of an epidemic may be due to the exhaustion of susceptible persons among the population. In the second and third place, either a loss of infectivity on the part of the organism or of susceptibility on the part of the population is necessary ' (Brownlee, ii. 244).

Brownlee goes on to remark that in any large solitary epidemic ' the symmetry of the course is an obvious and well-marked feature '. That the termination of an epidemic is not due to exhaustion of susceptible persons is apparent from the fact that, when an epidemic dies away, susceptibles are still present in the population at risk (op. cit., p. 248). Brownlee argued that the best explanation of the facts was afforded by the hypothesis that the infective power of the *materies morbi* decreased regularly from the beginning of the epidemic. He then pointed out that a law proposed by Farr and exhibited in detail by Evans would require the time-frequency curve to be of the ' normal ' type. It was found, however, that such a curve seldom adequately described the course of events. An explanation of this circumstance was suggested by the work of Pearson and Blakeman on random migration, and Brownlee modified the ' normal ' function by supposing that one of its constants, the standard deviation, itself varied. A law of frequency having been assumed for the standard deviation and the rate at which infective organisms are discharged being provisionally regarded as constant, a mathematical expression for the temporal law of an epidemic was deduced which can be fitted to the statistics by the method of moments (Brownlee, iii. 269–71). This curve is symmetrical and was found to fit certain epidemics better than the Pearson type curves applied to them by the author in his first study.

It is plain that Brownlee regards symmetry as a fundamental characteristic of the temporal curve of epidemics, although, of course, he recognizes that such a feature is not immediately apparent in many epidemics.

In actual records, indeed, symmetry is by no means an invariable nor even a frequent phenomenon in certain diseases. An examination of the numerous charts published in the Indian Plague Commission's Report on the seasonal prevalence of plague in India is alone sufficient to make this clear. If we take as an illustration the epidemic record of Bombay city, the epidemics of 1898, 1902, 1905, and 1906 appear to exhibit markedly different rates of increase and decrease. Ocular inspection of such charts is, however, a fallacious guide. I have studied the epidemic wave in the case of certain rural districts in more detail. The extreme asymmetry of the deaths curve, based upon all reported deaths for the districts of Amritsar and Gujrat has already been pointed out. A criticism which might be passed upon such material is that the data are collected over too wide an area to exhibit pure temporal effects and that the curve is complicated by the processes of inter-local spreading. Were the dissemination of plague to occur regularly from one or two centres, this would be a serious objection, but no such regular process can be discerned in the districts which have been specially studied.

Another method of examining the form of an epidemic time wave is to compare the numbers of villages which become infected for the first time in successive months, the number first infected in any month being some indication of the conditions favouring the prevalence of plague during that month. Table 12 and Diagram 5 contain the necessary information in the case of three Punjab districts and one district of the United Provinces. These also suggest that the conditions favouring epidemicity diminish at a rate other than that of their increase, although, of course, such evidence is of only minor importance. A consideration of all the facts does, however, seem to warrant the conclusion that in the Punjab villages the decline of the epidemic wave is more abrupt than its ascent and the Commission's other work affords a ready explanation of the facts. After a careful examination of data derived from numerous districts of India, the Commission concluded that ' in India it appears that a plague cannot exist in epidemic form in any of these places when the daily mean temperature is as high as 85° F. and over. If an epidemic is in progress, as soon as or very shortly after the temperature has reached the above height or even less, the plague epidemic receives a check and rapidly declines. While this is so, it would also appear that epidemics may come to an end when the temperature is most suitable. Another factor or factors must, therefore, be in operation in these instances ' (op. cit., p. 300).

We may reasonably attribute the asymmetry of the Punjab curves to the influence of the temperature or of something highly correlated therewith.

As an example of asymmetrical epidemics in which the decline is more gradual than the ascent, I may take the case of cholera. The epidemics at Hamburg in 1892 and St. Petersburg in 1909 are good illustrations. In Table 13 are contained the cases as recorded by Sticker (ii. 153) together with the equations and principal constants of the appropriate frequency

curves. It cannot, however, be said that the latter are satisfactory representations of the data.

It will appear that the functions which are appropriate to express the time changes in epidemics such as those considered cannot be perfectly symmetrical. Assuming for the moment that the basal phenomenon is a continuous change in infectivity on the part of the *materies morbi*, such that its expression is a symmetrical function of the time, then the curve is modified by the intervention of a factor which is in turn a function of temperature or something related to temperature. It is not difficult to invent mathematical expressions which would give effect to this train of thought, but as I have, so far, failed to deduce one which expresses the facts adequately, I do not propose to discuss the matter at length.

The one conclusion we seem justified in drawing is that the observed facts of temporal evolution during an epidemic are not, in the instances to which I have called attention, completely accounted for by an hypothesis which places the whole onus upon the changing characters of the infecting organism and neglects the environmental factors, the latter comprising effects not merely upon the population at risk and the *materies morbi* but also upon intermediate hosts and transmitters. It should, I think, be added that the work of Brownlee has introduced to the notice of epidemiologists a new and powerful instrument of research, since the laws propounded are, by the use of his method, capable of direct and systematic testing upon observed facts. In this way we can escape from the misty regions of pure theory in which those who speculate upon the course of epidemics are rather tempted to dwell.

I may refer more briefly to the factor scheduled under (*b*). That the resistance of the subject is variable has long been recognized, and in this connexion the problem of natural and acquired resistance has been often discussed. The bearing of this is, however, of more general than particular importance in connexion with the temporal evolution of an epidemic. Leaving on one side the instances in which the number of persons at risk is so greatly reduced by deaths or the acquirement of immunity through recovery from the disease that the chances of infection are notably reduced, the one casewhich has been elaborately examined is that of rat plague. The problem to be solved was whether the epizootic might be brought to an end by an increase in the proportion of immune to total rats. The general conclusion reached by the Indian Plague Commission was that ' there can be little doubt that an increase in the proportion of immune to susceptible rats as an epizootic progresses assists to some extent in bringing it to a close. In Poona no very direct evidence, however, was obtained in favour of this contention other than that, at the close of the epizootic in the city, an increasing number of rats were found which were either recovering or had actually recovered from the disease ' (*Journal of Hygiene*, x. 530).

In the case of the human subject, somewhat similar considerations apply. But when we are dealing with large centres of population, there

can be little doubt that the epidemic declines long before the susceptibles have been exhausted either by death or recovery.

(c) *Environmental factors.* We may, for purposes of discussion, classify the factors to be included in this group under three headings : (1) physiographical, (2) economic, (3) biological.

(1) *Physiographical.* Here fall to be considered the influence of climate and geological structure upon the course of an epidemic. I have already pointed out that, in the case of plague, temperature or something correlated therewith is of great importance in limiting the outbreak. But a consideration of all the facts points to the conclusion that this influence is indirect. If we fix our attention upon variations in temperature *within* the epidemic season, it seems impossible to show that they affect the rate of mortality. Thus, in the Punjab villages the plague season extends from November to July, and the great bulk of the deaths occur between the beginning of March and the middle of June. The severity of the outbreaks varies enormously in different years. A comparison of mortality statistics with records of rainfall or temperature from the district providing the deaths does not suggest any correspondence (see Greenwood, p. 129, Table XXXIX). The conclusion seems to be that such influence as is exerted by meteorological phenomena must be indirect, and the suggestion that it applies rather to the intermediate carriers of infection than to the organism or its human victims, is an obvious one.

Turning to the question of soil, it is well known that in the past considerable importance has been attached to the nature of the soil and its bearing on the disposal of products of cadaveric decomposition in connexion with epidemic plague. The ætiological theory which gave such discussions their importance is no longer widely accepted. There can be no doubt that, for instance, in the different districts of the Punjab the severity of contemporaneous outbreaks of plague varies very substantially from district to district. This will appear from the table (Table 14) which records the percentage mortalities in infected villages of five districts in three severe outbreaks. But it will be noticed that the order of severity is not maintained in different years, a fact which tells against the belief that these differences can be the expression of a permanent force dependent upon soil conditions. Again even more marked differences are observed intra-locally. Thus in the Gujrat epidemic of 1906–7, it was possible to mark out an area, included within a circle with a radius of ten miles, the rate of mortality in the affected villages of which was more than double that observed in other infected villages (Greenwood, p. 86). These facts indicate the caution necessary in assigning reasons for local differences in the severity of an epidemic, but that such differences have to be taken into account is plain. It will have been noticed in the table that the Gujrat and Rohtak districts occupy the first and second place in the scale of severity, and it may also be mentioned that the intra-local divergencies within these districts were more marked than elsewhere.

(2) *Economic Factors.* An extensive study of village plague, involving

the consideration of thousands of villages seemed to me to justify the following conclusions: (*a*) Large villages are more liable to become infected than small ones, but (*b*) small villages, when infected, exhibit a higher proportional mortality than do large ones. The second of these rules is subject to exceptions, but applies to the majority of cases. It seemed to be difficult to account for this result without resort to the hypothesis that the prosperity, type of house, and occupations of the inhabitants are correlated with the absolute size of the community. Evidence on these points has not yet been collected and analysed. That economic circumstances may be of paramount importance in determining the origin of any infectious disease, will hardly be questioned. It is also reasonable to suppose that the same conditions will affect the severity. Whether, however, such effect will be manifested in a mere change of scale or whether the *form* of the epidemic time curve will be modified, are questions which cannot be answered without fuller information than we possess.

Under this heading should, perhaps, also be ranked the effects of sanitary administration, but it will be convenient to reserve what I desire to say upon these until we have noted the *Biological Factors*.

A study of the biological factors extrinsic to the subject and object of the epidemic really resolves itself into an examination of the bionomics of such living creatures as are implicated in the transmission of the disease from person to person during an epidemic. It is unnecessary to remind the section that this branch of epidemiology has, perhaps, attracted more attention and been the object of more study than any other during the last few years. The bearing of such researches upon our immediate problem is direct. Thus environmental factors, which may not seem of importance when we confine our attention to the subject and object of an epidemic disease, may profoundly modify the life cycle of an intermediate host, and the time curve of the epidemic may exhibit the effects of such modification. In this connexion I would refer to the investigations of Niven and Dudfield dealing with epidemic diarrhœa and the recent paper of C. J. Martin.

Having now, briefly and inadequately, passed in review some of the factors which may profitably be discussed on the present occasion, it may be fitting to conclude with a reference to that aspect of epidemiology which almost alone interests those without the ranks of our profession. I mean the effect of immediate human action upon the course of an epidemic. It is in dealing with this matter that the risk of separating the immediate from the secular aspects of epidemic disease becomes most serious. One is prone to argue that because in a certain year an epidemic of a given disease endured longer and was more fatal than an epidemic of the same disease in a later year, therefore the measures adopted in the second case had necessarily a share in the result. If in order to avoid this fallacy of time we compare simultaneous outbreaks in different countries, the question of geographical and climatic variations is begged. A comparison restricted to within a small area will also be insufficient owing

to the fact that sanitary administration tends more and more to be standardized within the jurisdiction of one and the same central government.

That these are not mere *a priori* criticisms has been abundantly demonstrated by my colleague Professor Sticker, many of whose pages are melancholy records of misplaced self-satisfaction and premature self-congratulation on the part of administrators and sanitarians. The general reflections suggested by a knowledge of such facts are, I think, as follows.

In the event of an epidemic disease being generated by localized conditions of whatever order, it seems plain that our chances of checking its spread must vary directly as the state of sanitary organization. It may, however, often be true that the rapidity of epidemic evolution transcends any practicable rate of organizing counter-measures. The latter consideration does not imply a council of despair or warrant the suggestion that a highly-developed sanitary organization is useless in controlling an epidemic disease. Consequently any one who doubts whether our means of controlling epidemic disease be so efficient as the general public imagine, must not be regarded as an advocate of sanitary reaction. It is, however, permissible to suggest that the measures, at present, it must be confessed, indefinite enough in character, generally associated with the so-called eugenic movement may ultimately develop into a far more important system for the control of future epidemics than any *ad hoc* actions whatsoever.

If I may adopt a somewhat threadbare metaphor, our attitude towards epidemic disease seems rather too much that of army administrators concerting plans for the mobilization of an army to cope with foreign invaders or domestic rebels. Perhaps it might be wiser to seek to remove the conditions which provoke wars and rebellions. Sanitarians have long striven to do so in the actual case. But we might also seek to eliminate the potential rebels and chauvinists. The former plan is the ideal of nearly all medical publicists, it is the ideal of the present. The latter plan is the eugenic ideal and may be the goal of the future. If it be true that an inherited susceptibility exists not only in the case of such diseases as tuberculosis, but also in the case of exanthems, the eugenic ideal would be worthy of consideration, and in striving to attain it we should influence the course of epidemics very effectually by diminishing the proportion of the race likely to fall victims to them. These are matters which we, as epidemiologists, must carefully consider, recognizing that they are neither lightly to be accepted nor lightly to be dismissed with a reference to the results of laboratory experiment or clinical observation—both valuable methods of inquiry, but neither entitled to claim immunity from critical examination.

I must conclude, as I began, by expressing my sense of the inadequacy with which I have treated the important subject set down for discussion, my regret being only mitigated by the thought that such a paper as this may serve as a stimulus to the utterances of more competent scholars and thinkers, if only in virtue of the many crudities and errors it contains.

Conclusions

1. The immediate course in time of an epidemic is a phenomenon of sufficient regularity to suggest that laws descriptive of it may ultimately be propounded.

2. One factor in the evolution of an epidemic is likely to be found in changes taking place in the infecting organism as a consequence of which the latter's power of infecting susceptible subjects is a function of the time.

3. No law at present enunciated adequately describes the evolution in time of all epidemics.

4. In the case of certain diseases it is probable that the influence of such extrinsic factors as temperature, or something highly correlated with temperature, is of great importance in determining the course of an epidemic.

5. The effect of intervention *ab extra* on the course of epidemics is uncertain.

6. It is impossible to predict the course of any given epidemic without careful consideration of the previous history of the disease in the country under consideration and in the world at large.

Note A.—On the Fitting of Frequency Curves to Statistics

The figures recording the number of cases of, e. g., an epidemic disease reported in successive weeks are subject to errors, partly errors of observation, partly the result of what we generally call the effects of chance. In the hope of arriving at a knowledge of the fundamental law, the effects of which are partly hidden by these extraneous circumstances, the statistician substitutes for the irregular figures or their graph a curve which is the locus of some mathematical equation. Several methods have been proposed, that of Pearson being the one usually adopted. It is assumed that if the diagram which results from plotting the number of cases as ordinates and the time intervals as abscissæ warrants us in supposing that the frequency has but one maximum, i. e., that there are not two or more peaks to the curve (regard being had to the number of cases available for study) and that it rises from and falls to zero, then the slope of the curve may be represented by an equation of the form : $\frac{dy}{dx}=\frac{y\ (x+a)}{F\ (x)}$, a being a constant. It is then assumed that $F\ (x)$ can be expanded by Maclaurin's theorem so that the equation becomes

$$\frac{1}{y}\frac{dy}{dx}=\frac{x+a}{b_0+b_1x+b_2x^2\ldots}$$

where b_0, b_1, b_2, &c., are constants.

The constants are then obtained by equating the moments of the curve to the actual moments of the statistics, i. e., equations such as

$$N\mu'_n=\int yx^n.dx$$

are formed, the left-hand side being computed from the actual data. If no moments beyond the fourth be employed (the n^{th} moment of a set of statistics about a fixed point is the sum of the n^{th} powers of the distances of each point on the base line from the fixed point, each multiplied by the value of the ordinate at that point), a family of curves is obtained which has been found in practice capable of describing many kinds of distribution. Full details of the method are to be found in the following memoirs and text-books:

Pearson, K.—'Skew Variation in Homogeneous Material,' *Phil. Trans. A.*, clxxxvi, 343, and cxcvii, 443.

—— *On the General Theory of Skew Correlation and Non-Linear Regression*, London, 1905 (Dulau), pp. 3–9.

Elderton, W. Palin.—*Frequency Curves and Correlation*, London (C. and E. Layton), pp. 1–105.

Blaschke, E.—*Vorlesungen über mathematische Statistik*, Leipzig, 1906 (Teubner), pp. 164–7 and 202–8.

Note B.—On the Analysis of Epidemics by the Method of Differences

In an appendix to the second edition of his work on the prevention of malaria, Sir Ronald Ross explains in detail a method he has devised for the mathematical study of epidemics. The fundamental problems reduce to the two following:

(1) If it be supposed that the population of a given area is being continuously changed by birth, death, immigration, and emigration, and if further some event (i.e. the development of a disease) happens to a constant proportion per unit of time, then let the problem be to determine what proportion of the whole will, after the expiration of a given time, have been affected by the happening and to what proportions the event will have happened, once, twice, ..n times.

(2) Is the same problem modified by the additional supposition that a constant proportion of the affected class revert to the unaffected class per unit of time?

If we divide our 'universe' into the mutually exclusive groups (*a*) affected, and (*b*) not affected, it is clear that an evaluation of the transfers from one group to the other as time passes will depend upon the solution of a system of finite difference or of differential equations, in accordance with whether the unit chosen be finite or infinitesimal.

Sir Ronald Ross obtains and solves the equations which he applies to the special case of malaria. The results in that particular case are of great importance, but, apart from this, it is evident that the method might be applied to the study of other diseases. Some difficulties may be encountered in practice. Thus Sir Ronald considers (p. 678) the case in which the infection rate varies directly as the number of infected persons present at a given moment. It might be necessary in some cases to regard the rate as a more complex function of such number, with the

result that difficulty might be experienced in integrating our equations. It often happens, however, that *a priori* objections of this kind do not prove of importance in practice, and it is to be hoped that other epidemiologists will avail themselves of Sir Ronald Ross's methods. Sir Ronald's fame as an investigator should ensure attention on the part of others to this field of inquiry.

REFERENCES

(Extensive bibliographies will be found in the works marked *.)

Ballard, E.—'On the Prevalence and Fatality of Scarlatina as influenced by Sex, Age, and Season,' *Trans. Ep dem. Soc.*, N. S., 1887, vii. 183.

Berndt, F. A. C.—*Die Scharlachfieber-Epidemie im Custrinschen Kreise in den Jahren* 1817–19, &c., Leipzig und Berlin, 1820.

Brownlee, J.—(1) 'Statistical Studies in Immunity; the Theory of an Epidemic,' *Proc. Roy. Soc. Edin.*, 1906, xxvi. 484.

—— (2) 'Certain Considerations on the Causation and Course of Epidemics,' *Proc. Roy. Soc. Med., Epidem. Sect.*, June, 1909.

—— (3) 'The Mathematical Theory of Random Migration and Epidemic Distribution,' *Proc. Roy. Soc. Edin.*, 1911, xxxi. 262.

*Creighton, C.—*A History of Epidemics in Britain*, Cambridge, 1894.

Dudfield, R.—'Diarrhœa in 1911,' *Proc. Roy. Soc. Med., Epidem. Sect.*, 1912, p. 99.

Evans, G. H.—'On Some Arithmetical Questions involved in the Rise and Progress of Epidemics,' *Trans. Epidem. Soc.*, 1873–5, iii. 551.

Farr, W.—*Vital Statistics*, London, 1885.

Fothergill, J.—*An Account of the Sore Throat attended with Ulcers*, &c., London, 1748.

Graves, R. J.—*Clinical Lectures on the Practice of Medicine*, London, 2nd edition, 1848.

Greenwood, M., jun.—'On Some of the Factors which Influence the Prevalence of Plague,' *Journ. Hygiene*, 1911, xi. 62.

*Haeser, H.—*Lehrbuch d. Geschichte d. Medicin u. d. epid. Krankh.*, Jena, 1882.

Hippocrates.—*Fuchs's Translation*, München, 1895–1900. Van der Linden's text and Latin translation, Leiden, 1665.

Heiberg, P.—'Die Letalität des Scharlachfiebers in Dänemark während der Jahre 1850–1900,' *Zeitschr. f. Hygiene*, 1910, lxvi. 291.

*Hirsch, A.—*Handbook of Geographical and Historical Pathology*, Creighton's translation, London, 1883.

Longstaff, G. B.—'On Some Statistical Indications of a Relationship between Scarlatina, Puerperal Fever, and certain other Diseases,' *Trans. Epidem. Soc.*, 1879–80, iv. 421.

Martin, C. J.—'Insect Porters of Bacterial Infections,' *Brit. Med. Journ.*, 1913, i. 1.

*Most, G. F.—*Versuch einer kritischen Bearbeitung der Geschichte des Scharlachfiebers*, Leipzig, 1826.

Müller, R.—'Künstliche Erzeugung neuer vererbbaren Eigenschaften bei Bacterien,' *Münch. med. Wochenschr.*, 1909, p. 885.

Murphy, Sir Shirley F.—'Seasonal Variations in Age Distribution and Fatality,' *Trans. Epidem. Soc.*, N. S., 1897, xvii. 116.

—— 'Some Points in the Decline of the Birth- and Death-rates,' *Journ. Roy. Sanitary Inst.*, 1912, xxxiii. 337.

Nash, J. T. C.—'Evolution in Relation to Disease,' *Trans. Epidem. Soc.*, N. S., 1906, xxv. 204.

Niven, J.—'Summer Diarrhœa and Enteric Fever,' *Proc. Roy. Soc. Med., Epidem. Sect.*, 1910, p. 131.

Pearson, K., and Blakeman, J.—*A Mathematical Theory of Random Migration*, London, 1906 (Dulau).

Penfold, W. J.—'Studies in Bacterial Variation,' *Journ. Hyg.*, 1911, xi. 30.

—— 'On the Specificity of Bacterial Variation,' *Journ. Hyg.*, 1912, xii. 195.

Plague Investigations in India, Reports on, Reprinted from the Journal of Hygiene, 2 vols., Cambridge, 1911.

Prinzing, F.—*Handbuch der medizinischen Statistik*, Jena, 1906.

Ransome, A.—'On the Relations between Diphtheria and Scarlet Fever,' *Trans. Epidem. Soc.*, 1870–5, iii. 520.

*Richter, P.—'Beiträge zur Geschichte des Scharlachs,' *Arch. f. Geschichte d. Med.*, 1908, i. 161.

Ross, Sir Ronald.—*Prevention of Malaria*, 2nd edition, London, 1911 (Murray).

Rumsey, H. jun.—'An Account of an Epidemic Sore Throat which appeared at Chesham in Bucks, in the year 1788,' *London Med. Journ.*, 1789, x. 7.

Schnitzlein, E.—*Das Scharlachfieber, seine Geschichte, Erkenntniss und Heilung*, München, 1859.

*Sticker, G.—*Abhandlungen a. d. Seuchengeschichte u. Seuchenlehre*, I. Band (Pest), Giessen, 1908–10; II. Band (Cholera), Giessen, 1912.

Sydenham, T.—*Opera Omnia*, ed. Greenhill, London, 1844.

Watt, R.—*Treatise on the History, &c., of Chin Cough, &c., to which is subjoined an Inquiry into the Relative Mortality of the Principal Diseases of Children, &c.*, Glasgow, 1813.

Wedemeier, J. T.—*Historia Scarlatinae nuper Gottingae grassatae*, Gottingen, 1785.

Whitelegge, A.—'Age, Sex, and Season in Relation to Scarlet Fever,' *Trans. Epidem. Soc.*, N. S., 1887, vii. 153.

Willan, R.—*Miscellaneous Works*, London, 1821.

Wilson, J. T.—'A Contribution to the Natural History of Scarlet Fever,' *Public Health*, February 1897.

Withering, W.—*An Account of the Scarlet Fever and Sore Throat at Birmingham*, 1779.

Zierl, L.—*De Scarlatinae et Morbillorum Epidemia anno* 1818 *et* 19 *observata*, Landishuti, 1819.

Zimmermann, J. G.—*Dissertatio de Scarlatina in annis* 1775 *et* 6 *epidemica* (in J. P. Frank, *Delectus opusc. medicorum*, ii. 253, Ticini, 1786).

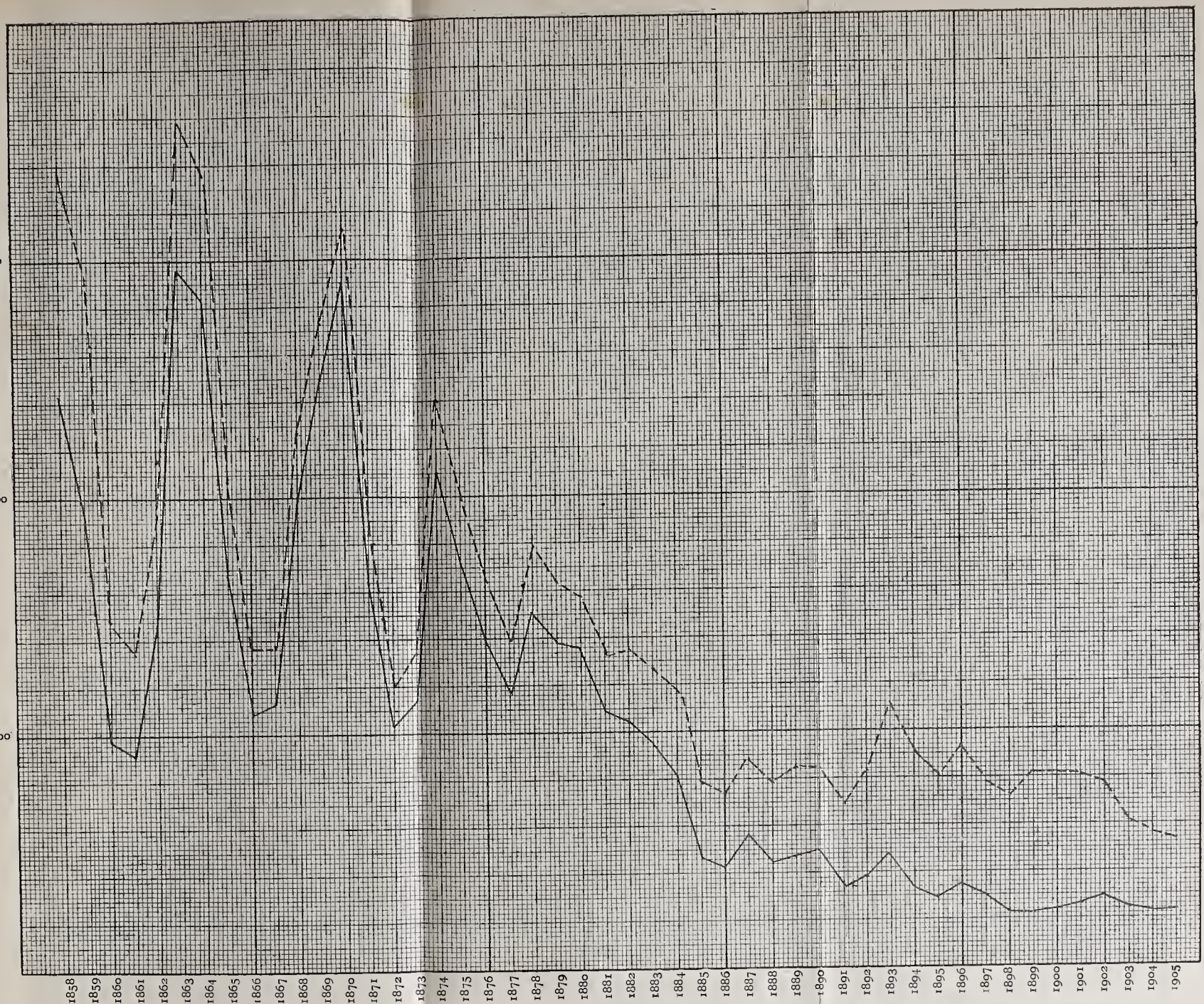

DIAGRAM 1. England and Wales. Scarlet Fever; deaths per million, 1858–1905 (continuous line). Scarlet Fever and Diphtheria; deaths per million, 1858–1905 (broken line).

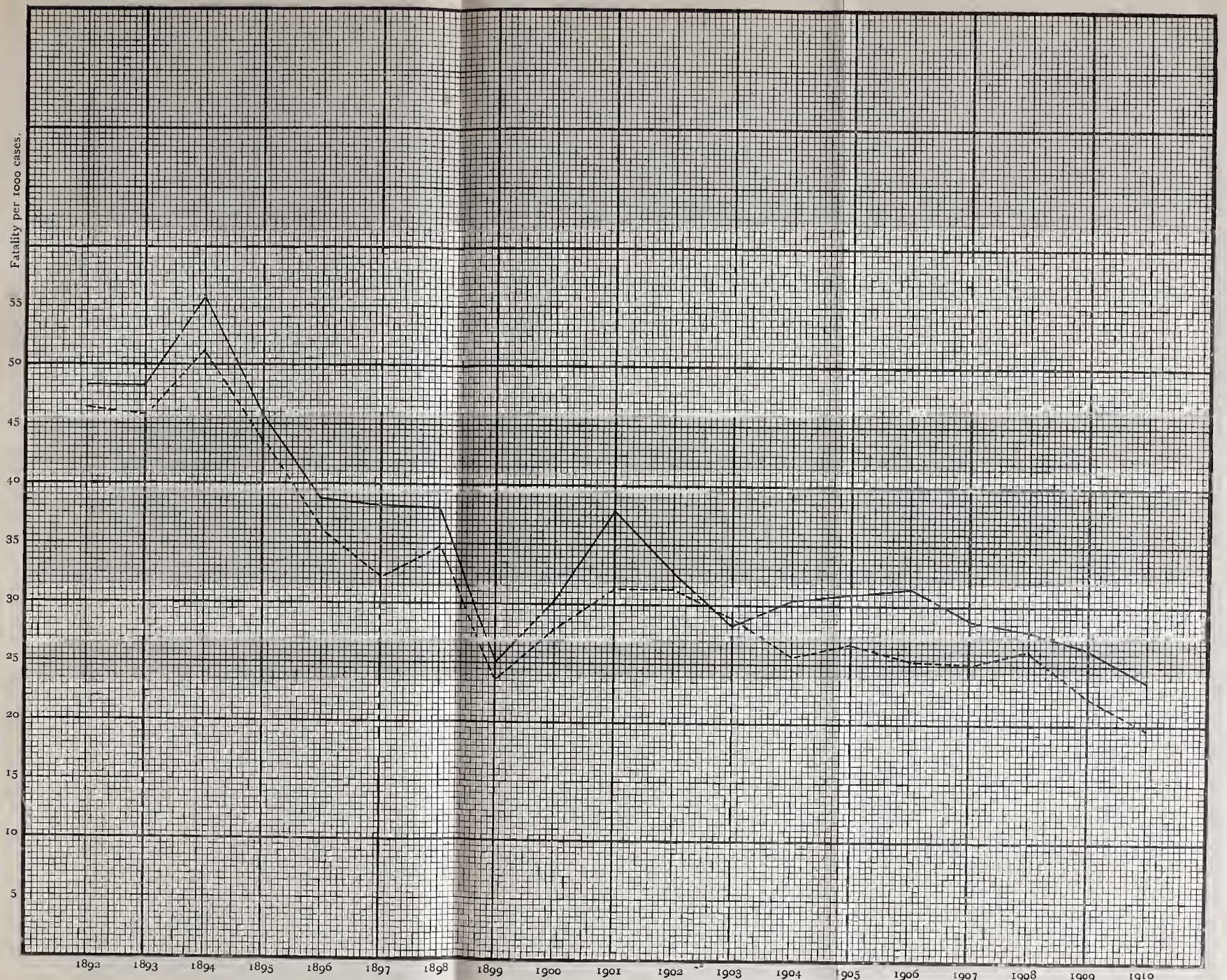

DIAGRAM 2. Scarlet Fever. London fatality rates, 1892–1910 (corrected). Males continuous; females dotted line.

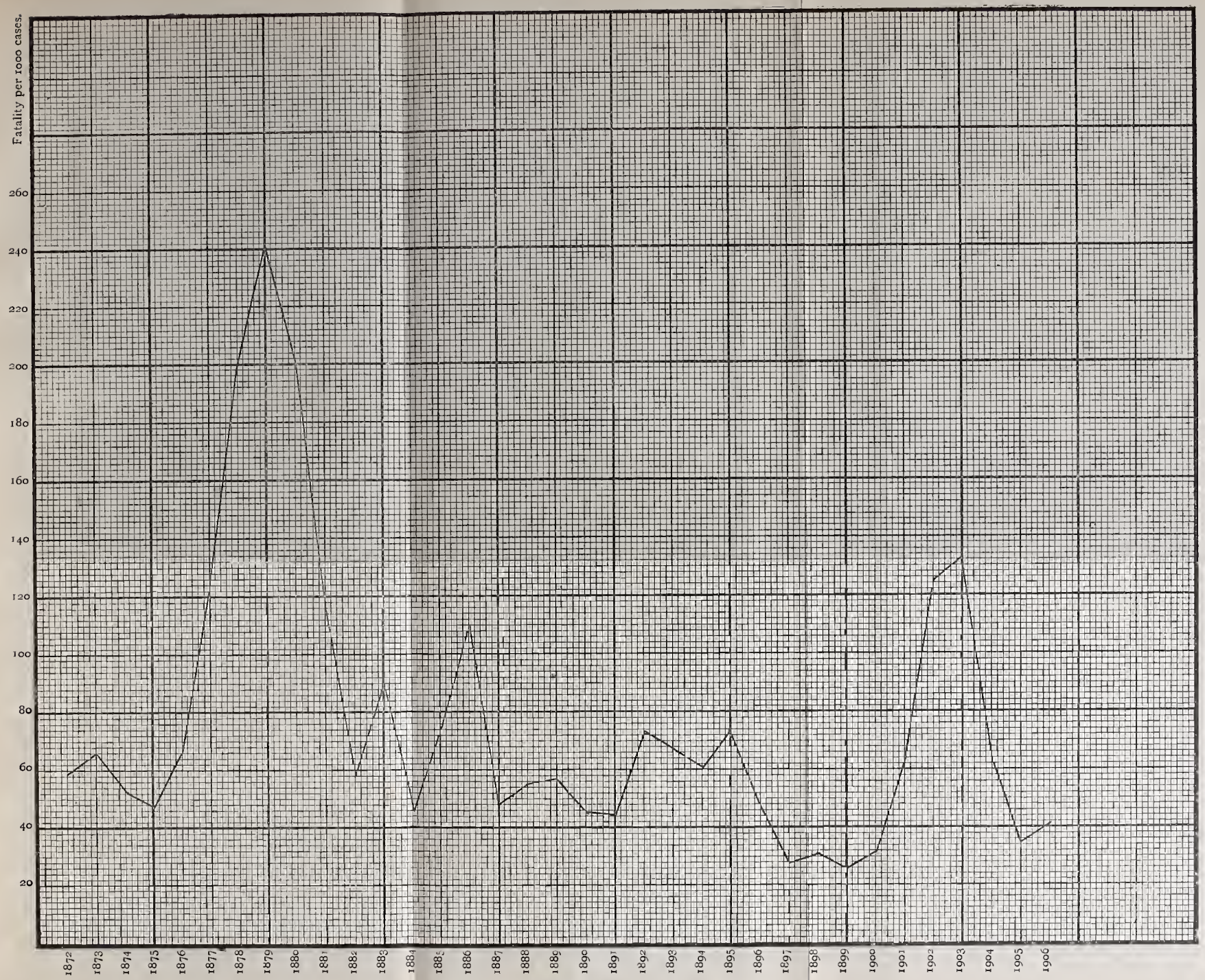

DIAGRAM 3. Fatality of Scarlet Fever, Hamburg, 1872–1906.

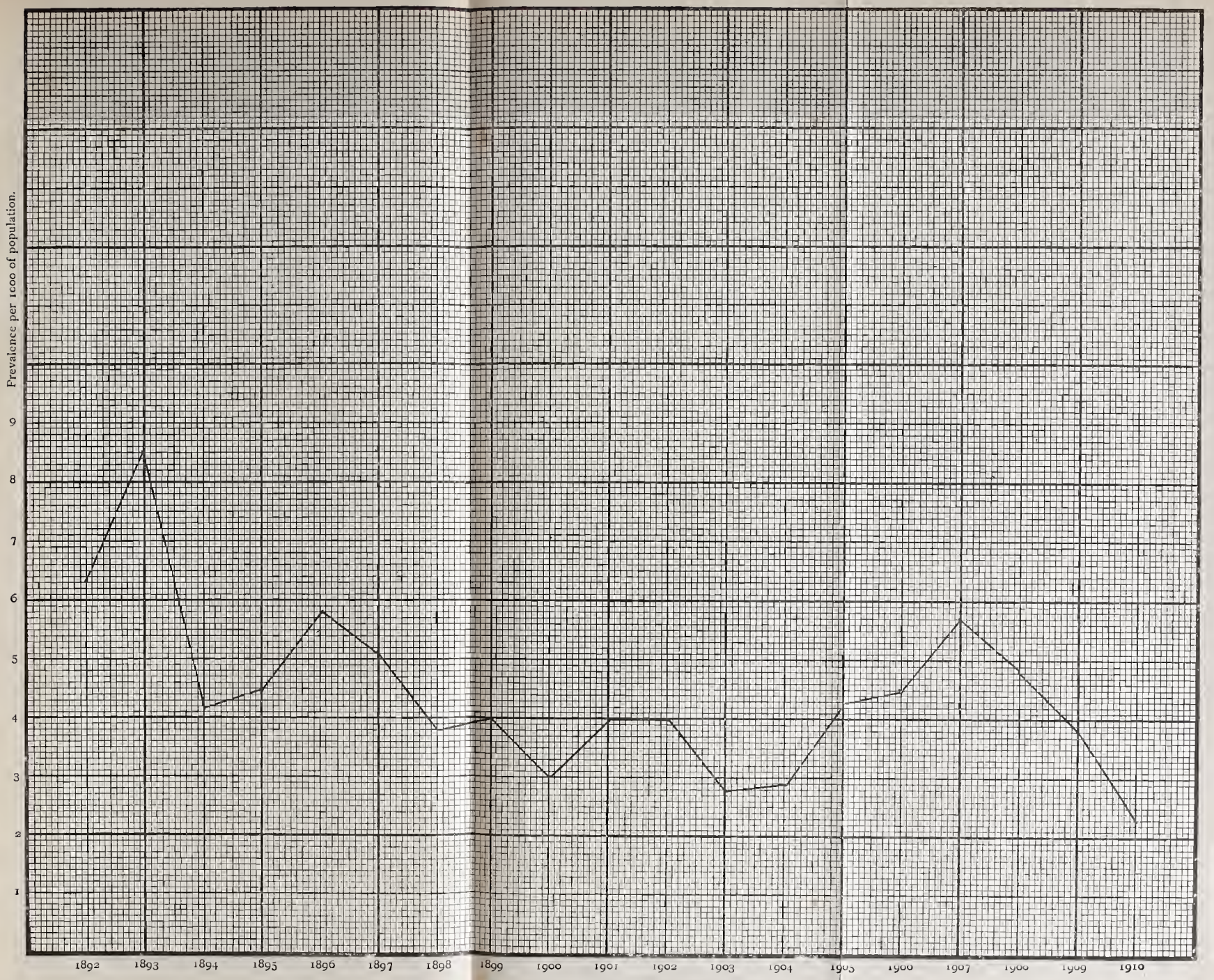

DIAGRAM 4. Prevalence of Scarlet Fever, London, 1892-1910.

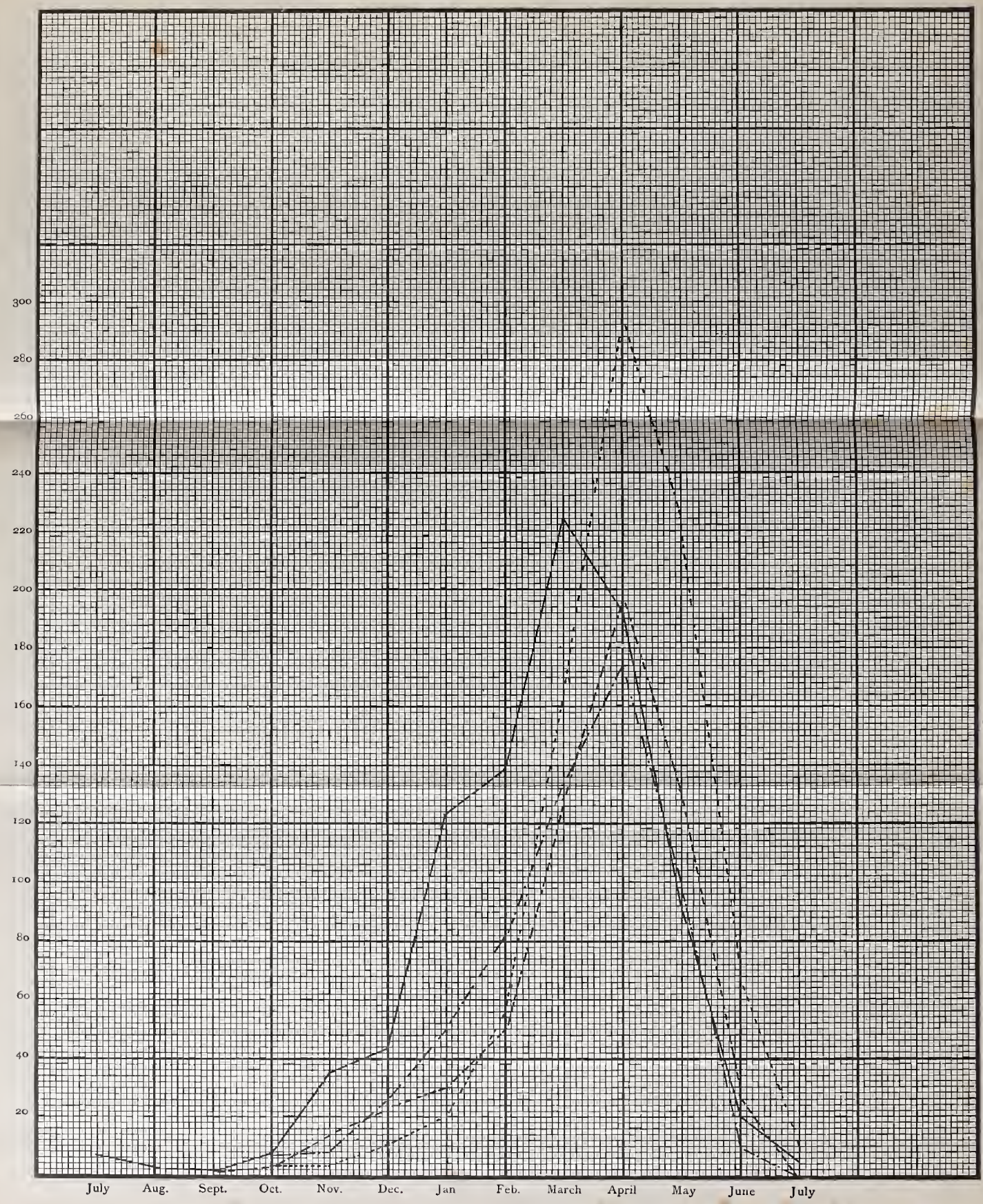

Diagram 5 Frequency of villages ranged in order of month of first infection, 1906-7.
Hoshiapur, continuous line. Amritsar – – – – Mozaffarnagur — · — · — · Gujrat - - - - - -

TABLE 3

County of London

Scarlet Fever Corrected Fatality (1892–1910)

Rates per 1000

Males.*

	0–	1–	2–	3–	4–	5–	10–	15–	20–	25–	35–	45–	55 *up.*	*All Ages.*
1892	1·88	5·39	10·37	9·82	6·38	11·55	1·33	·63	·07	·21	·25	·10	·13	48·11
1893	1·75	5·45	9·21	8·83	6·19	12·88	1·60	·87	·46	·76	—	·06	·10	48·16
1894	1·77	7·48	9·75	9·49	9·19	14·14	1·89	·62	·61	·67	·17	—	—	55·78
1895	2·23	5·45	9·31	8·18	6·52	10·80	1·78	·25	·59	·28	—	·22	·13	45·74
1896	1·64	5·14	6·88	8·00	4·83	8·71	1·81	·87	·33	·20	·09	·17	—	38·67
1897	1·61	4·48	9·02	7·34	3·85	7·79	1·49	·70	1·16	·40	·39	—	—	38·23
1898	1·21	5·97	8·69	6·72	3·94	7·70	1·47	·29	·67	·77	·13	·38	·13	38·07
1899	1·92	2·42	4·72	4·97	2·33	5·60	1·20	·23	·57	·85	·14	—	·10	25·05
1900	1·98	3·31	5·72	5·04	6·00	4·73	1·59	·55	·56	·61	·13	—	—	30·22
1901	1·84	4·62	7·79	6·61	3·49	10·59	·98	·74	·38	·36	·33	·12	—	37·85
1902	1·46	4·15	7·53	5·79	3·71	7·63	1·02	·70	·32	·37	—	—	—	32·68
1903	1·07	3·61	5·14	5·13	3·65	7·76	·45	·67	·59	·36	—	—	—	28·43
1904	1·07	4·63	6·74	4·81	2·83	7·07	1·99	·52	·17	·59	—	—	—	30·42
1905	1·52	5·14	7·28	4·96	2·69	6·89	1·04	·45	·24	·45	·27	—	—	30·93
1906	·75	4·14	5·50	6·73	3·93	7·15	1·47	·68	·63	·14	·13	—	—	31·25
1907	1·56	2·72	5·74	5·57	3·08	6·70	1·29	·62	·47	·36	·44	·14	—	28·69
1908	1·08	2·46	4·90	5·69	3·59	6·38	1·50	·89	·39	·56	·31	·17	—	27·92
1909	1·63	3·21	3·50	3·97	3·69	7·14	1·36	·79	·37	·41	—	·20	—	26·27
1910	·80	3·21	4·21	4·01	2·81	5·81	1·80	·80	·40	·20	—	·20	—	23·65

Females.*

	0–	1–	2–	3–	4–	5–	10–	15–	20–	25–	35–	45–	55 *up.*	*All Ages.*
1892	1·65	6·47	10·27	9·06	6·19	9·81	1·44	·26	·23	·75	·20	—	—	46·33
1893	1·44	6·21	9·28	7·70	6·96	9·98	2·13	·68	·54	·52	·18	·14	·13	45·89
1894	2·22	6·30	8·88	9·46	6·30	13·64	2·24	·53	·82	·58	·21	—	—	51·18
1895	1·63	5·59	10·30	8·78	4·81	9·69	1·28	·31	·80	·28	—	—	·13	43·60
1896	1·89	4·60	8·24	6·70	4·35	8·28	·80	·39	·57	·26	·15	—	—	36·23
1897	1·76	4·16	6·12	7·10	3·61	7·51	·93	·30	·24	·31	·07	·21	·08	32·40
1898	1·36	5·24	7·36	6·12	5·22	7·25	1·13	·30	·19	·34	·12	·17	—	34·80
1899	·67	3·60	5·64	4·19	2·70	4·87	·69	·25	·40	·55	—	·15	—	23·71
1900	1·22	4·77	5·87	5·05	2·84	6·38	·54	·29	·25	·39	—	·12	—	27·72
1901	1·50	4·16	7·51	6·20	2·85	5·41	1·81	·60	·49	·32	·18	·14	·13	31·30
1902	1·60	4·09	6·37	5·83	2·61	8·19	·95	·32	·59	·53	—	—	·09	31·17
1903	1·50	3·13	5·43	5·69	2·75	8·18	·73	·66	·32	·47	—	—	—	28·86
1904	1·60	3·81	5·40	4·00	3·21	5·60	1·31	—	·38	·22	—	·22	—	25·75
1905	1·58	3·78	5·52	4·04	3·10	6·37	·94	·31	·46	·52	·27	—	—	26·89
1906	·94	3·74	5·38	4·28	2·45	5·77	1·58	·51	·28	·30	·08	—	—	25·31
1907	1·30	3·36	5·68	4·25	3·45	4·91	·86	·68	·26	·29	·21	—	—	25·25
1908	1·01	4·32	4·88	4·37	3·06	6·21	1·09	·40	·30	·46	·17	—	—	26·27
1909	·82	4·03	2·62	4·00	2·94	5·89	1·48	·22	·26	·14	—	—	—	22·40
1910	1·59	1·44	5·16	2·00	2·22	5·70	1·14	·19	·15	·24	—	—	·10	19·93

* The figures at each age-group were obtained by multiplying $\frac{\text{no. of deaths}}{\text{no. of cases}}$ by $\frac{100\,d}{D}$, where d = no. of cases at that age in the Standard Population and D=total number of cases in the Standard Population (Males, 1910, formed the Standard Population).

TABLE 4 A

County of London

Scarlet Fever Fatality (1892–1910)

Males.

	0–		1–		2–		3–		4–		5–		10–		15–		20–		25–		35–		45–		55 *up.*		*All ages.*	
	Cases.	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*
1892	230	36	531	76	846	110	1134	110	1233	75	4891	135	2285	22	797	11	364	1	257	2	76	3	21	1	6	1	12671	583
1893	281	41	726	105	1161	134	1501	131	1610	95	6727	207	3187	37	1353	26	533	9	455	13	128	—	34	1	8	1	17704	800
1894	177	26	393	78	663	81	832	78	821	72	3551	120	1386	19	438	6	221	5	198	5	38	1	14	—	5	—	8737	491
1895	183	34	387	56	677	79	866	70	964	60	3683	95	1704	22	538	3	231	5	192	2	52	—	9	1	6	1	9492	428
1896	220	30	491	67	893	77	1176	93	1193	55	4659	97	2201	29	725	14	331	4	266	2	75	1	23	2	8	—	12261	471
1897	187	25	471	56	778	88	1075	78	1145	42	4191	78	1761	19	645	10	256	11	201	3	49	3	18	—	6	—	10783	413
1898	119	12	341	54	578	63	783	52	877	33	3207	59	1214	13	466	3	201	5	174	5	50	1	16	3	6	1	8032	304
1899	119	19	296	19	608	36	855	42	945	21	3286	44	1376	12	582	3	239	5	189	6	47	1	14	—	8	1	8564	209
1900	79	13	262	23	460	33	622	31	629	36	2300	26	1125	13	495	6	288	6	218	5	51	1	16	—	3	—	6548	193
1892–1900	1595	236	3898	534	6664	701	8844	685	9417	489	36495	861	16239	186	6039	82	2664	51	2150	43	566	11	165	8	56	5	94792	3892
1901	144	22	351	43	645	63	796	52	889	27	3279	83	1399	10	669	11	358	5	225	3	59	3	17	1	5	—	8836	323
1902	132	16	363	40	657	62	874	50	962	34	3292	60	1345	10	586	9	336	4	215	3	58	—	11	—	4	—	8835	288
1903	90	8	303	29	497	32	592	30	689	24	2264	42	912	3	340	5	182	4	149	2	32	—	5	—	4	—	6059	179
1904	90	8	285	35	509	43	652	31	740	20	2664	45	831	12	259	3	155	1	135	3	34	—	10	—	1	—	6365	201
1905	142	18	359	49	668	61	980	48	1090	28	3828	63	1196	9	401	4	228	2	179	3	47	2	9	—	4	—	9131	287
1906	129	8	364	40	667	46	962	64	1041	39	3978	68	1503	16	468	7	256	6	194	1	50	1	10	—	5	—	9627	296
1907	139	18	526	38	806	58	1163	64	1327	39	4935	79	1811	17	656	9	343	6	300	4	73	5	14	1	1	—	12094	338
1908	134	12	429	28	700	43	925	52	1110	38	4327	66	1653	18	511	10	275	4	237	5	62	3	24	2	5	—	10392	281
1909	111	15	305	26	593	26	739	29	795	28	3342	57	1416	14	461	8	220	3	197	3	50	—	5	1	4	—	8238	210
1910	60	4	188	16	398	21	505	20	523	14	2088	29	687	9	226	1	135	2	133	1	32	—	10	1	4	—	4989	118
1901–1910	1171	129	3473	344	6140	455	8188	440	9166	291	33997	592	12753	118	4577	67	2488	37	1964	28	497	14	115	6	37	—	84566	2521

TABLE 4 B

County of London

Scarlet Fever Fatality (1892–1910)

Females.

	0–		1–		2–		3–		4–		5–		10–		15–		20–		25–		35–		45–		55 *up.*		*All ages.*	
	Cases.	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*	*Cases*	*Deaths.*	*Cases.*	*Deaths.*	*Cases.*	*Deaths.*
1892	212	29	454	78	816	105	1184	106	1253	74	5889	138	2672	28	870	5	470	4	425	12	127	4	40	—	10	—	14422	583
1893	284	34	655	108	1135	132	1485	113	1671	111	7467	178	3618	56	1270	19	703	14	669	13	178	5	43	3	19	3	19197	789
1894	141	26	419	70	665	74	877	82	932	56	3865	126	1661	27	516	6	263	8	274	6	62	2	17	—	11	—	9703	483
1895	162	22	371	55	635	82	888	77	981	45	4105	95	1830	17	589	4	305	9	283	3	91	—	19	—	6	1	10265	410
1896	185	29	483	59	891	92	1179	78	1228	51	5305	105	2409	14	691	6	430	9	418	4	125	3	31	—	11	—	13386	450
1897	185	27	435	48	821	63	1084	76	1221	42	4847	87	2065	14	604	4	343	3	342	4	89	1	19	2	10	1	12065	372
1898	115	13	309	43	542	50	810	49	943	47	3522	61	1580	13	446	3	282	2	238	3	53	1	12	1	10	—	8862	286
1899	90	5	314	30	523	37	822	34	932	24	3782	44	1797	9	545	3	338	5	293	6	73	—	13	1	5	—	9527	198
1900	69	7	229	29	435	32	642	32	628	17	2820	43	1278	5	469	3	328	3	272	4	65	—	17	1	1	—	7253	176
1892–1900	1443	192	3669	520	6463	667	8971	647	9789	467	41602	877	18910	183	6000	53	3462	57	3214	55	863	16	211	8	83	5	104680	3747
1901	104	13	299	33	595	56	832	51	922	25	3634	47	1594	21	677	9	444	8	337	4	73	2	28	2	6	1	9545	272
1902	98	13	313	34	626	50	886	51	965	24	3680	72	1454	10	566	4	366	8	355	7	77	—	22	—	9	1	9417	274
1903	80	10	265	22	441	30	623	35	724	19	2508	49	939	5	344	5	253	3	225	4	57	—	10	—	3	—	6472	182
1904	75	10	257	26	443	30	709	28	750	23	2988	40	949	9	323	—	284	4	241	2	45	—	9	1	1	—	7074	173
1905	122	16	379	38	694	48	927	37	1081	32	4404	67	1615	11	432	3	296	5	310	6	47	2	14	—	9	—	10330	265
1906	90	7	353	35	667	45	899	38	1071	25	4497	62	1741	20	537	6	391	4	353	4	80	1	19	—	4	—	10702	247
1907	130	14	438	39	828	59	1166	49	1306	43	5795	68	2398	15	670	10	523	5	460	5	93	3	16	—	8	—	13831	310
1908	131	11	349	40	654	40	904	39	1131	33	4921	73	2022	16	561	5	452	5	406	7	115	3	24	—	10	—	11680	272
1909	88	6	290	31	549	18	734	29	819	23	3836	54	1485	16	411	2	311	3	378	2	78	—	22	—	15	—	9016	184
1910	53	7	183	7	294	19	455	9	567	12	2422	33	845	7	236	1	177	1	218	2	55	—	7	—	8	1	5520	99
1901–1910	971	107	3126	305	5791	395	8135	366	9336	259	38685	565	15042	130	4757	45	3497	46	3283	43	720	11	171	3	73	3	93587	2278

TABLE 5

Administrative County of London

Diphtheria Fatality (1892–1910)

	No. of Cases.			*Crude Fatality Rates %*			*Corrected Fatality Rates.*	
Year.	*Males.*	*Females.*	*Total.*	*Males.*	*Females.*	*Total.*	*Males.*	*Females.*
1892			8368			22·22		
1893	6165	7529	13694	26·42	21·81	23·89	26·25	26·48
1894	4987	6203	11190	25·81	23·07	24·29	23·25	24·78
1895	5107	6116	11223	22·68	19·59	20·99	19·96	20·82
1896	6183	7625	13808	20·98	17·97	19·32	18·52	19·12
1897	5915	7277	13192	18·48	16·34	17·30	16·84	16·88
1898	5454	6401	11855	16·65	13·72	15·07	15·02	14·36
1899	6211	7472	13683	14·97	13·87	14·37	13·21	14·00
1900	5569	6416	11985	14·08	12·64	13·31	12·29	12·89
1901	5657	6499	12156	12·11	10·31	11·15	11·04	10·75
1902	4975	5756	10731	11·42	10·35	10·85	10·27	10·49
1903	3613	4125	7738	9·66	10·01	9·85	8·19	9·59
1904	3340	3879	7219	10·75	9·93	10·31	8·86	9·39
1905	3023	3459	6482	9·13	8·24	8·66	7·43	7·63
1906	3721	4324	8045	9·59	8·35	8·93	8·08	8·15
1907	4007	4764	8771	9·81	8·54	9·12	8·00	8·04
1908	3711	4291	8002	9·84	8·46	9·10	8·25	8·05
1909	3110	3569	6679	10·06	8·80	9·39	7·86	8·29
1910	2592	2902	5494	8·49	7·82	8·14	7·18	7·45

TABLE 6

Berlin

Scarlet Fever Fatality (1903–1909)

Year.	*Scarlet Fever Cases.*	*Deaths.*	*No. of Cases not notified before Death.*
1903	1351	341	152
1904	1817	425	190
1905	1306	428	166
1906	2017	285	69
1907	2160	179	38
1908	3531	276	55
1909	6559	627	148

TABLE 7

Hamburg

Bericht des Medizinalrates über die medizinische Statistik des Hamburgischen Staates.

Scharlach, 1872–1906

Year.	*Rate %*	*Year.*	*Rate %*	*Year.*	*Rate %*
1872	5·9	1883	9·0	1894	6·1
1873	6·6	1884	4·6	1895	7·3
1874	5·3	1885	7·5	1896	4·7
1875	4·8	1886	11·0	1897	2·8
1876	6·7	1887	4·8	1898	3·1
1877	12·4	1888	5·5	1899	2·6
1878	19·8	1889	5·7	1900	3·2
1879	24·1	1890	4·6	1901	6·3
1880	20·2	1891	4·5	1902	12·5
1881	11·7	1892	7·3	1903	13·3
1882	5·9	1893	6·7	1904	6·3
				1905	3·5
				1906	4·1

TABLE 8

Scarlet Fever, London, 1892–1910

Cases per 1000 of population (all ages)

Year.	*Cases per* 1000 *Population.*	
1892	6·3	
1893	8·5	5·9
1894	4·2	
1895	4·5	
1896	5·8	
1897	5·1	
1898	3·8	4·7
1899	4·0	
1900	3·0	
1901	4·0	
1902	4·0	3·5
1903	2·7	
1904	2·9	
1905	4·3	
1906	4·5	4·3
1907	5·7	
1908	4·8	
1909	3·8	3·4
1910	2·3	

TABLE 9

Scarlet Fever, London, 1892–1910

Incidence per 1000 at certain ages

Year.	*Males.*			*Females.*		
	0–5	5–10	10–15	0–5	5–10	10–15
1892	16	22	11	15	26	13
1893	21	30	15	21	33	17
1894	12	16	7	12	17	8
1895	12	16	8	12	18	9
1896	16	21	11	16	23	11
1897	15	19	9	15	21	10
1898	11	14	6	11	15	8
1899	11	14	7	11	17	9
1900	8	10	5	8	12	6
1901	11	15	7	11	16	8
1902	12	15	7	12	17	7
1903	9	10	4	9	11	4
1904	9	12	4	9	13	5
1905	13	17	6	13	20	8
1906	13	18	7	12	20	8
1907	16	22	9	16	26	11
1908	13	20	8	13	22	10
1909	10	15	7	10	17	7
1910	7	10	3	6	11	4

TABLE 10

Scarlet Fever Fatality 1893–1910, Metropolitan Borough of Poplar (Dr. Alexander's Report for 1910)

Year.	*Cases.*	*Deaths.*	*Fatality per* 1000.
1893	1794	100	56
1894	795	42	53
1895	1002	47	47
1896	946	34	36
1897	1100	28	25
1898	638	22	34
1899	423	9	21
1900	499	7	14
1901	559	14	25
1902	673	21	31
1903	449	20	45
1904	549	24	44
1905	991	28	28
1906	571	12	21
1907	1283	43	34
1908	1279	36	28
1909	727	22	30
1910	471	12	25

TABLE 11

England and Wales

Deaths per 1,000,000 *from various Infectious Diseases* (1901–9) *among children aged* 0–5

Year.	*Scarlet Fever.*	*Whooping Cough.*	*Measles.*	*Diphtheria and Croup.*	*All Causes.*
1901	710	2650	2260	1640	54130
1902	780	2530	3200	1400	49070
1903	660	2430	2260	1070	47290
1904	600	3000	2970	1000	51620
1905	590	2170	2660	940	44660
1906	510	2040	2240	1000	45270
1907	480	2480	2930	900	40890
1908	400	2360	1830	820	40570
1909	460	1700	2850	780	36780
		Relative Values			
1901	1·312	4·896	4·175	3·030	100·000
1902	1·590	5·166	6·521	2·853	100·000
1903	1·396	5·139	4·779	2·263	100·000
1904	1·162	5·812	5·754	1·937	100·000
1905	1·321	4·859	5·956	2·105	100·000
1906	1·127	4·506	4·948	2·209	100·000
1907	1·174	6·065	7·166	2·201	100·000
1908	·986	5·817	4·511	2·021	100·000
1909	1·251	4·622	7·749	2·121	100·000
		Deaths per 1,000,000 (10-year periods)			
1861–70	4644	3782	3011	2371	68600
1871–80	3504	3667	2579	1525	63379
1881–99	1667	3366	3127	1595	56789
1891-1900	844	3086	3247	1719	57736
*1901–9	577	2373	2578	1061	45587
		Relative Values			
1861-70	6·770	5·513	4·389	3·456	100·000
1871-80	5·529	5·786	4·069	2·406	100·000
1881-90	2 937	5 930	5·509	2·810	100·000
1891-1900	1·462	5·345	5·624	2·977	100·000
*1901-9	1·266	5·173	5·619	2·313	100·000

* Average of 9 years.

TABLE 12

Numbers of Villages First Reporting Plague Deaths in Various Months

Month.	*Hoshiapur* 1906–7.	*Amritsar* 1906–7.	*Mozaffarnagar* 1906–7.	*Gujrat* 1906–7.
July	7	—	—	—
August	3	—	—	—
Sept.	2	2	—	—
Oct.	8	3	7	3
Nov.	35	14	8	3
Dec.	44	23	26	10
Jan.	123	30	50	20
Feb.	139	50	81	56
March	224	128	133	165
April	192	197	174	294
May	90	129	99	224
June	20	27	10	67
July	5	—	—	9

TABLE 13

Epidemic Cholera

Hamburg, 1892

Week.	*Cases.*
1	477
2	5092
3	5587
4	2568
5	1908
6	927
7	303
8	69
9	39
10	8
11	1
12	2
	16981

Constants of Distribution.

$\beta_1 = 1{\cdot}0243$
$\beta_2 = 4{\cdot}0978$
$\kappa_2 = -1{\cdot}104$
Skewness = ·67223
Mean = 3·29397
Mode = 2·36155

Equation $y = 5567{\cdot}9 \left(1 + \frac{x}{1{\cdot}2281}\right)^{\cdot 95412} \left(1 - \frac{x}{14{\cdot}4499}\right)^{11{\cdot}2259}$

Origin at Mode.

St. Petersburg, 1909

Week.	*Cases.*
1	197
2	1456
3	2568
4	1535
5	794
6	418
7	257
8	126
9	103
10	105
11	70
12	0
	7629

Constants of Distribution.

$\beta_1 = 2{\cdot}3315$
$\beta_2 = 5{\cdot}6192$
$\kappa_2 = -1{\cdot}5927$
Skewness = 1·2886
Mean = 3·7993

Equation $y = {\cdot}000105\, x^{-\cdot 15497} (17{\cdot}7999 - x)^{5{\cdot}97097}$

Origin at 1·8749

TABLE 14

Epidemic Plague

District.	*Percentage Mortalities in Infected Villages.*		
	1903–4	1904–5	1906–7
Gujrat	10·1	4·6	9·5
Rohtak	—	6·0	7·0
Amritsar	3·7	4·4	3·8
Mozaffarnagar . . .	2·4	3·2	6·7
Hoshiapur	4·6	3·0	2·6

OXFORD: HORACE HART
PRINTER TO THE UNIVERSITY